crHIV-1 super-infection of I_T cells

HIV-1 *nef* downregulates CD4 thus not allowing for HIV-1 superinfection.
But crHIV-1 can infect a cell multiple times since it does not encode *nef*.
For simplicity, we have limited our consideration to the case of crHIV-1 dual infection
(i.e. superinfection of 2 crHIV-1).

We consider 2 variations of super-infection:

Model 2 where superinfection produces a different species of dually infected cell, a cell that has altered P and D values.

Model 1 (superinfection leads to I_D cells)

$$\dot{T} = \square - d\,T - k\,V\,T - k\,V_T\,T$$

$$\dot{I} = k\,V\,T - \square\,I$$

$$\dot{I}_T = k\,V_T\,T - d\,I_T - k\,V\,I_T - k\,V_T\,I_T$$

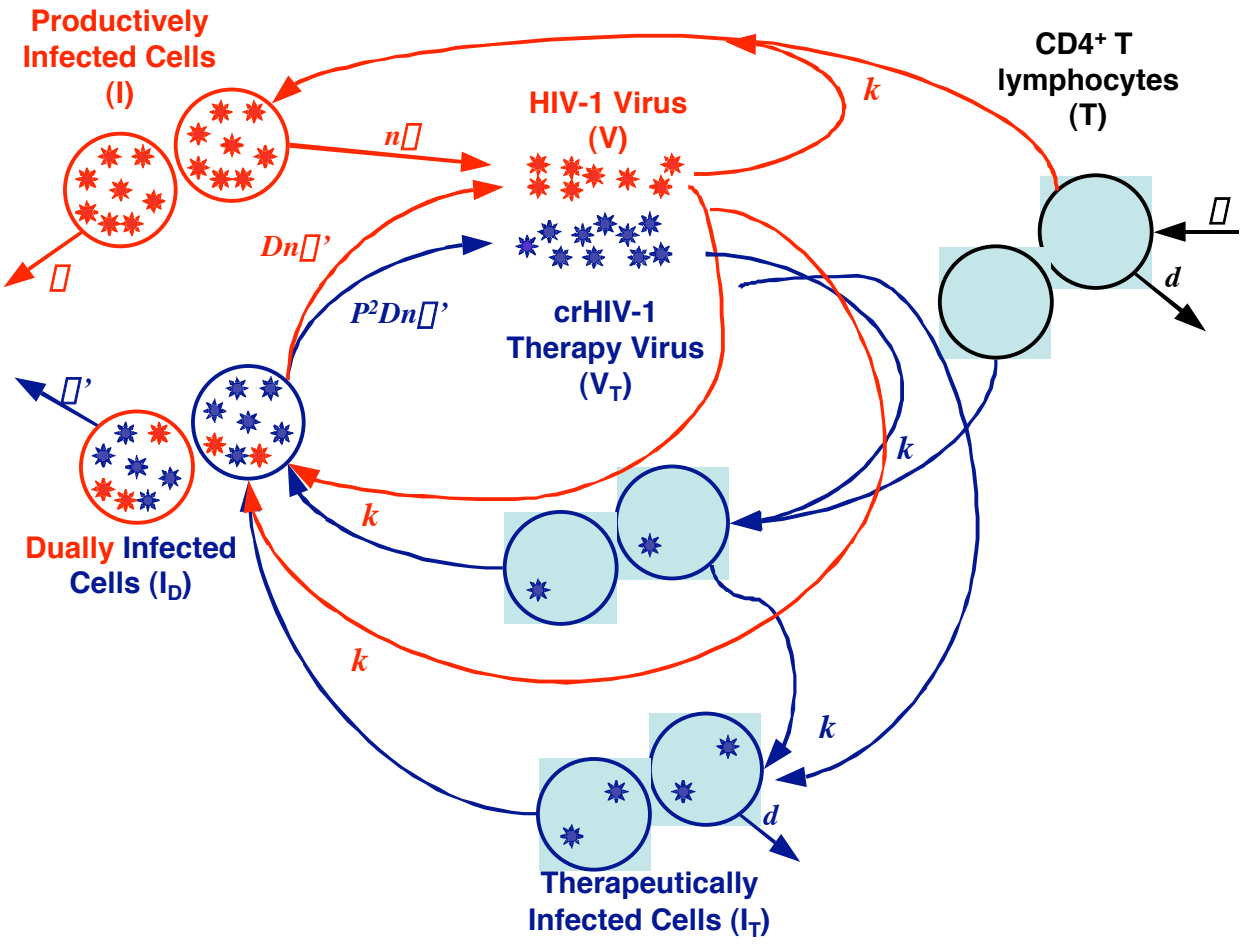
$$\dot{I}_{T2} = k\,V_T\,I_T - d\,I_{T2} - k\,V\,I_{T2}$$

$$\dot{I}_D = k\,V\,I_T + k\,V\,I_{T2} - \delta'\,I_D$$

$$\dot{V} = n\,\delta\,I + D\,n\,\delta'\,I_D - c\,V$$

$$\dot{V}_T = P^2\,D\,n\,\delta'\,I_D - c\,V_T$$

The schematic below describes Model 1.



Results from superinfection Model 1 are identical to Eqs. **1-6**.

Model 2

(superinfection produces a different species of
dually infected cell, a cell that has altered P and D values)

$$\dot{T} = \square - d\,T - k\,V\,T - k\,V_T\,T$$

$$\dot{I} = k\,V\,T - \square\,I$$

$$\dot{I}_T = k\,V_T\,T - d\,I_T - k\,V\,I_T - k\,V_T\,I_T$$

$$\dot{I}_{T2} = k\,V_T\,I_T - d\,I_{T2} - k\,V\,I_{T2}$$

$$\dot{I}_D = k\,V\,I_T - \delta'\,I_D$$

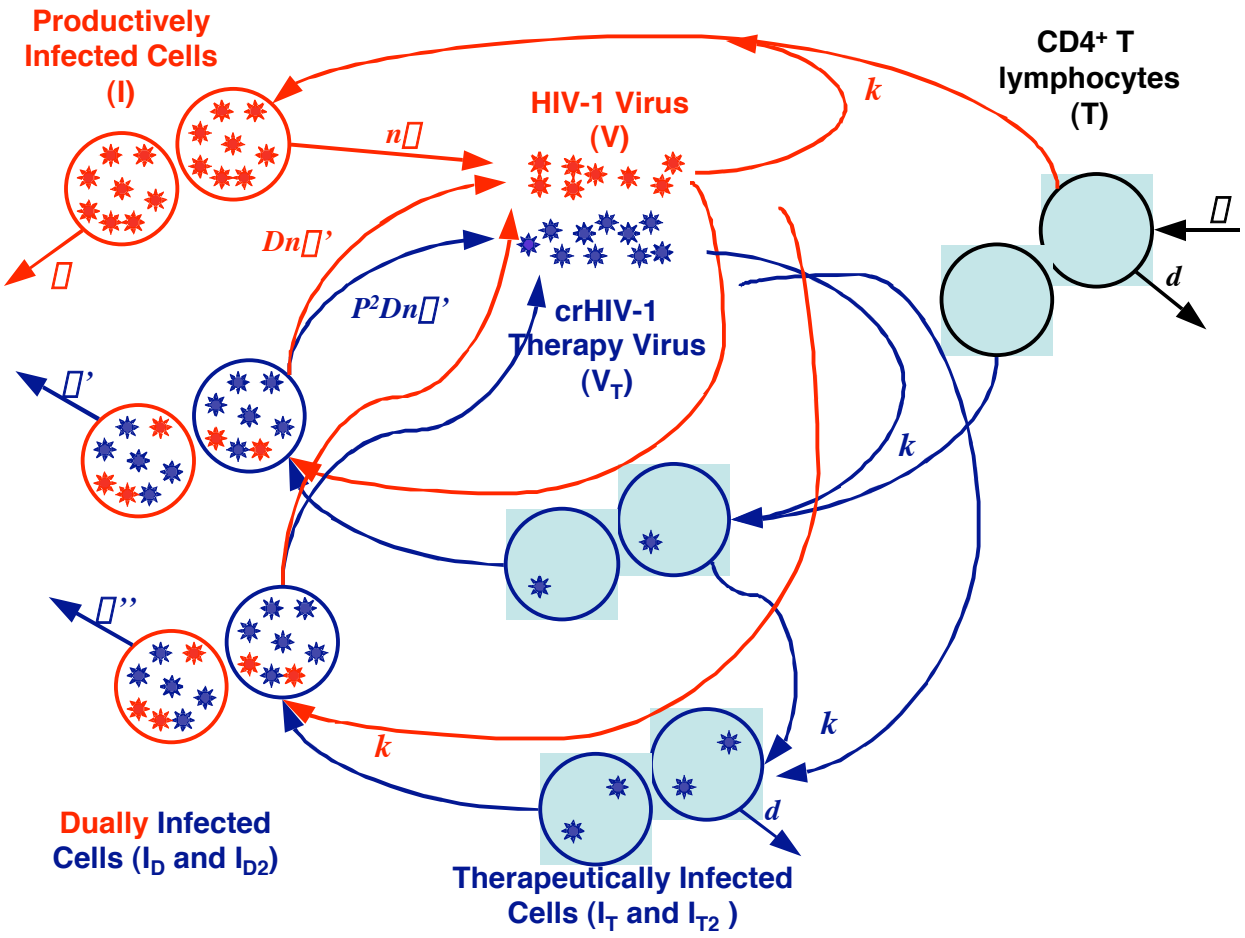
$$\dot{I}_{D2} = k\,V\,I_{T2} - \delta''\,I_D$$

$$\dot{V} = n\,\delta\,I + D\,n\,\delta'\,I_D + D_2\ n\ \delta''\,I_{D2} - c\,V$$

$$\dot{V}_T = P^2\,D\,n\,\delta'\,I_D + (2\,P)^2\,D_2\,n\,\delta''\,I_{D2} - c\,V_T$$

since there are 2 copies of the crHIV–1
genome in I_{D2} cells
$P \to 2\,P$
and
$D \to D_2$
where
$D \le D_2 \le 2\,D$ and $\delta'' = D_2\,\square$

The schematic below describes Model 2.



Results from superinfection Model 2 show significantly improved HIV-1 set point reduction compared to Eqs. **1-6**.
Thus, crHIV-1 superinfection, which is likely to occur, improves therapy and Eqs. **1-6** appear to be a lower limit of therapeutic efficacy.